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#31	Search Lu HH and HCV	12:38:29	3
#30	Search Lu HH	12:38:21	104
#27	Search Selby M and HCV	12:35:31	12
#26	Search Selby M	12:35:24	94
#25	Search PKR and HCV subgenomic	12:34:53	6
#23	Search PKR and HCV	12:34:39	81
#24	Search PKR and HCV replicon	12:33:52	9
#21	Search Dubensky 1996 and sindibis virus	12:31:37	2
#19	Search Behrens 1998 and pestivirus	12:30:30	2
#18	Search Behrens 1998	12:30:18	123
#17	Search Behrens 1998 and HCV	12:30:09	0
#14	Search HBsAg and ayw1/ayw2	07:18:48	11
#10	Search HBV and ayw1/ayw2 variant	07:17:58	0
#9	Search HBsAg and ayw1/ayw2 variant	07:17:52	0
#8	Search HBV ayw1/ayw2 variant	07:17:41	0
#3	Search hbv and sudan	07:16:08	6
#2	Search HBV and sudan	07:16:06	0
#1	Search HBV 29681 strain	07:15:57	0

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#10 Search **PKR knock out** Limits: **Publication Date to 2000/08/04**

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#9 Search **PKR knock out and virus infection** Limits: **Publication Date to 2000/08/04**

12:50:31 0

#2 Search **PKR mutant and virus infection** Field: **All Fields,** Limits: **Publication Date to 2000/08/04**

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#1 Search **PKR mutant and virus infection**

12:46:00 32

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(FILE 'HOME' ENTERED AT 13:05:59 ON 05 JAN 2006)

FILE 'CAPLUS' ENTERED AT 13:06:07 ON 05 JAN 2006

L1	210	"HOUSE KEEPING GENE"
L2	2055	"ANTI-VIRAL"
L3	1	L1 AND L2
L4	13	MUTATION AND L1
L5	51207	LETHAL
L6	0	L1 AND L5
L7	285870	MUTATION
L8	13	L1 AND L7
L9	2663	IL2
L10	1	L1 AND L2
L11	0	INF GENE MUTATION
L12	51207	LETHAL
L13	32950	GENE (W) MUTATION
L14	537	L12 AND L13
L15	65045	CELL (S) DEATH
L16	9	L14 AND L15
L17	2	L1 AND L15
L18	0	IL2 KNOCK OUT MICE
L19	2663	IL2
L20	6972	KNOCK OUT
L21	2	L19 AND L20
L22	977	IL8
L23	0	L22 AND L20
L24	0	L20 AND L22
L25	3858	CCR5
L26	2	L25 AND L20
L27	53	IL8 RECEPTOR
L28	0	L27 AND L20
L29	51	INTERLEUKINE
L30	0	L29 AND L20
L31	164691	CYTOKINE
L32	340	L31 AND L20
L33	137349	DEATH
L34	24	L33 AND L32

FILE 'CAPLUS, BIOSIS' ENTERED AT 11:10:04 ON 05 JAN 2006

L1 144 "ANTI VIRAL RESPONSE"
L2 11387 KNOCK (W) OUT
L3 0 L1 AND L2
L4 7 PKR AND L1
L5 203666 VIRUS (S) INFECTION
L6 2 L4 AND L5
L7 0 HCV AND L4
L8 297 PKR (S) MUTANT
L9 90 INFECTION AND L8
L10 1097807 VIRUS
L11 82 L9 AND L10
L12 2 HCV AND L11

FILE 'STNGUIDE' ENTERED AT 11:22:11 ON 05 JAN 2006

L13 0 CELL AND L11
L14 0 TRANSFECTED

FILE 'CAPLUS, BIOSIS' ENTERED AT 11:27:02 ON 05 JAN 2006

L15 6770361 CELL
L16 79 L15 AND L11
L17 79 INFECTION AND L16
L18 7412 REPLICON
L19 0 L18 AND L17
L20 0 TRANSFECTION AND L19
L21 1 HCV AND L17
L22 39 "DOMINANT NEGATIVE PKR"
L23 12 L17 AND L22

FILE 'STNGUIDE' ENTERED AT 11:31:21 ON 05 JAN 2006

ACCESSION NUMBER: 2002:428614 CAPLUS
DOCUMENT NUMBER: 137:5026
TITLE: NOG immunodeficient mouse for disease models and human antibody production
INVENTOR(S): Ito, Mamoru; Kobayashi, Kimio; Nakahata, Tatsutoshi; Tsuji, Koichiro; Habu, Sonoko; Koyanagi, Yoshio; Yamamoto, Naoki; Sugamura, Kazuo; Ando, Kiyoshi
PATENT ASSIGNEE(S): Central Institute for Experimental Animals, Japan
SOURCE: PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002043477	A1	20020606	WO 2001-JP9401	20011025
W: CA, JP, US				
RW: AT, BE, CH, PT, SE, TR	CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,			
CA 2402459	AA	20020606	CA 2001-2402459	20011025
EP 1338198	A1	20030827	EP 2001-978918	20011025
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003182671	A1	20030925	US 2002-221549	20020910
PRIORITY APPLN. INFO.:			JP 2000-367296	A 20001201
			WO 2001-JP9401	W 20011025

AB An immunodeficient mouse is established by cross intercross of NOG/Shi mouse, SCID mouse, and IL-2Ry **knock-out** mouse (NOG mouse). The NOG mouse shows functional deficiency of T and B lymphocytes, NK cells, macrophages, and dendritic cells. Transplantation of human stem cells to NOG mouse shows engagement of the cells without rejection. It is useful in constructing human antibody, stem cell assay system, disease models and drug screening for leukemia and AIDS.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2002:411839 CAPLUS

DOCUMENT NUMBER: 137:76241

TITLE: Caspases - their role in apoptosis and other physiological processes as revealed by **knock-out** studies

AUTHOR(S): Sadowski-Debbing, Kenneth; Coy, Johannes F.; Mier, Walter; Hug, Hubert; Los, Marek

CORPORATE SOURCE: Clinic for Craniomaxillofacial Surgery, Ahaus, D-48683, Germany

SOURCE: Archivum Immunologiae et Therapiae Experimentalis (2002), 50(1), 19-34

CODEN: AITEAT; ISSN: 0004-069X

PUBLISHER: Ossolineum Publishing House

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review with 113 refs. Caspases are crucial mediators of apoptosis, a form of physiol. cell **death**. Their activation is carefully controlled by a phylogenetically conserved **death** program, which is indispensable for the homeostasis and development of higher organisms. Dysregulation of apoptosis contributes to the pathogenesis of many human diseases. As effectors of the apoptotic machinery, caspases are considered potential therapeutic targets. In vitro studies have demonstrated the requirement of caspase activity for both the triggering phase as well as the execution of apoptosis, thus providing a mol. base for the fine-tuning of this process by pharmacol. agents. The precise roles of the individual caspases in vivo and their functional relation to each other have been best demonstrated in genetically modified animals. The generation of single caspase-deficient mice have confirmed most of the data obtained in vitro and exposed some new aspects previously undetected in the cell culture system. Interestingly, inactivation of many caspases revealed not only their expected participation in apoptotic events as well as in the maturation of **cytokines**, but also provided hints about the role of at least some caspases in cell differentiation and stimulatory responses. Here, the authors discuss what these studies have unveiled about the role of individual caspases in development, apoptosis, and inflammation, with particular focus on their role beyond the apoptotic process.